# Candidate 3 evidence

3- depression

a) Persistent depressive disorder is similar to major depressive disorder in terms of symptoms but is less severe than major depressive disorder, according to the DSM-5 model to be diagmnosed with persistent depressive disorder you need to show 5 symptoms of depression on majority of days for 2 years, these symtpoms can inclkude insomnia/ hypersomnia, suicidial thoughts that can lead to self harming and attempts, low mood constantly where the person does have motivation to do simple tasks, a loss of interest in activities the person enjoyed before feeling depressed, and lack of social engagement with other people, it can also include moody behaviour towards other people, ofcourse these vary inm their extremes, other symptoms can icnlude using drugs to cope with thoughts. Low mood and dissociative behaviour is one the biggest indicators of depression and is one of trhe main symptoms you need to be diagnosed, although the ilness is less severe than major depression it is still debilitating to live with every day. The lack of socialising leads to a cycle of loneliness and makes things worse, persistent depressives are given bioligical treatments and cognitive behavioural therapy if needed.

b) According to the diathesis stress model, both biology and psychology and enviroment affect the chance of getting depression, it is widely respected bioloiigcal theory because it takes into account more than one factor that can lead to depression, as humans are very complex beings and it is unlikely one small thing can lead depression in every case this makes the approach more holistic. The diathesis part of the model refers to the idea that someone may have a genetic predisposition such as a mutated gene to depression or high neurotiscm, the diathesis can even be a traumatic psychological event that causes the person to have a predisposition to getting depression, the stress part of the model refers to external stressors that can lead to depression occuring in someone such as stressful life event, like divorce. Both of these together are crucial to developing depression,

the idea is that one cannot happen without the other. Otherwise the person will unlikely develop depression. The theory can actuallyt be evidenced somewhat by Caspi et al (2003), capsi studied gene differences that can cause depression but also measured life events over a longitudinal study. Caspi used 3 groups of people one, with the two short alleles of the 5HTT gene (gene related to serotonin ptroduction in the brain- according to the role of neurochemistry serotonin lacking can cause depression as serotonin changes mood). Group 2 had one short and one long allele, and group 3 had 2 short alleles of the gene, caspi found with his participants who where studies from age 3 to age 26 that those who had atleast one short allele of the 5HTT gene and atleast 4 or more stressful life events, 33% where elgible to be diagnosed with clinical depression, in comparison to those who had two long alleles but still 4 or more stressful life events, 17% where eligble to be diagnosed weith clinical depression. This therefore backs up the diathesis stress model somewhat as it shows that in more cases the genetic factor and the stressful life events are what leads to major depression. Although the study could be seen as contradicting, because if the diathesis stress model is correct, then 100% of people with 4 or more stressful life vents and atleast 1 short allele would be eligible to be diagnoses with major depression, it suggests that the diuathesis stress model although seen as holisitic, is missing a few key points for example cognitive thinking processes that can lead to the development of depression. The diathesis stress also does not think about the fact that everyone percieves stressful life events to a different extent, some people are better at coping with stressful events and may not even view a traumatic experience as a 'big deal' whereras another person may view the same experience as extremely trauamtic and cause them to develop depression. The diathesis stress does not allow for cognitive thinking which is a proven factor that is related to depression, although the cognitive approach is more reductionist than the diathesis stress it still account for things that cause depression, we can see this through the use of the tretament cognitive behavioural therapy which focuses on treating negative thought processes through task like thought catching with dathesis stress does not consider. The fact CBT has great success just goes to show the cognitive apprioach has major applications. The cognitive triad and Ellises ABC model both see that the way we see ourself- if it is a negative self schema we will have negative thoghts, something similar about diathesis stress and the cognitive approach is that they look to early life events to seek an explation, if we see ourselves in a negative light we will degrade ourselves more which can lead to more depressive thoughts according to beck, negative shcemas maintain the nagtive triad, by havviing a negative view of onself, a negative view of the world and a negative view others about you. The ABC model by ellis believes that the activation event (similar to daithesis, the siatheiss can be a psycholiigcal event at a young age) leads to a belief about oneself. If the belief is negative it will cause negative consequences leading to more depressive thoughts leading to dwepression, however if looking at oinself in a positive light as a belief, for example failinhg a pre lim instead of givijng up trying harder, this will not lead to depressive thoughts. The diathesis model does not think of this factor which is very true, if a person experiences the stress, if they think of

the stress positively will they still develop depression or not? Although the cognitive approach is more simplisitc and does not allow for human thought to be complex and impulsive unlike the diathesis, it should still be accounted for as it is evidenced in therapy, for example alloy et al (1999) found thjose with positive thinking styles, only 1% of them had depression, those with negativer thinking styles 17% had depression, this therfore goes to show thought processes in a cognitive way do affect if we will get depression.

Biolical treatments also evidence the diathesis stress model, such as the use of SSRI or SNRI these block the serotinin reputake in the neurons so more serotonin is avaialbel in the brain to boost mood, SNRI also block the rewputake of norepinephrine which is also associated with mood, these have shown great success especially SSRI's which have shown to have 80% to 90% effectiveness when treated, this therefore shows diathesis stress can be backed up by bioloigical treatments because bioligical treatments have shown to be successful, this further shows that there is a biology to depression otherwise these treatments would not work. Another advmnatage of thediathesis stress model is the stress, those with the same genetic predispositions could eiuther get depression or not based on their liviung state, those with low incomes who struggle to get by will be more likely to get depressed, especially if they have the genetics for it. Biological treatmentsa have been around for the longest compared to CBT, this therefore shows that it is very widely accepted that there is a bioliigcal cause to depression. The TADS study in 2007 also further proved bioligical treatments where the most effective the most quickly qwith 61% effectiveness by 12 weeks in, this therefore shows that ther eis biology to depression which the diathesis strews sdimportantly accounts for which most approaches may not. However CBT showed to be kore effective at 12 weeks with 71%, shwoing cognitive thoughts also lead to depression, this seriuously weakens the diathesis stress models stance. It is clearly not holisiutc enough.